

The epidemiology of obesity: the size of the problem

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The epidemic of obesity took off from about 1980 and in almost all countries has been rising inexorably ever since. Only in 1997 did WHO accept that this was a major public health problem and, even then, there was no accepted method for monitoring the problem in children. It was soon evident, however, that the optimum population body mass index is about 21 and this is particularly true in Asia and Latin America where the populations are very prone to developing abdominal obesity, type 2 diabetes and hypertension. These features are now being increasingly linked to epigenetic programming of gene expression and body composition *in utero* and early childhood, both in terms of fat/lean tissue ratios and also in terms of organ size and metabolic pathway regulation. New Indian evidence suggests that insulin resistance at birth seems linked to low birth weight

and a higher proportion of body fat with selective B12 deficiency and abnormalities of one carbon pool metabolism potentially responsible and affecting 75% of Indians and many populations in the developing world. Biologically there are also adaptive biological mechanisms which limit weight loss after weight gain and thereby in part account for the continuing epidemic despite the widespread desire to slim. Logically, the burden of disease induced by inappropriate diets and widespread physical inactivity can be addressed by increasing physical activity (PA), but simply advocating more leisure time activity is unrealistic. Substantial changes in urban planning and diet are needed to counter the removal of any every day need for PA and the decades of misdirected food policies which with free market forces have induced our current 'toxic environment'. Counteracting this requires unusual policy initiatives.

Keywords: nutrition, obesity.

Introduction

It is only a decade ago that the problem of obesity was seen as a minor issue which concerned only a few endocrinologists intrigued by the manifestations of the condition. Their main concern was to attempt to identify the genetic component of the obesity arising in a few children and adults who put on substantial amounts of weight. Clinical management was also known to be frustrating for both patients and doctors; the major improvements achieved on weight loss were universally recognized but the frequent inability of patients to achieve and maintain effective weight loss

led to clinical frustration and the temptation to blame the individual for a lack of will power.

Now, however, we have a completely different perspective. Obesity is the concern of most, if not all, governments who now readily accept its complexity and major public health impact. Their main concern is now rapidly moving towards prevention and not simply treating the consequences. The medical profession, however, is working out in clinical and experimental terms what the principal mechanisms underlying weight gain and how to determine the complex intestinal/neurological control of energy

balance and the mechanism of biological resistance to weight loss in obese patients.

The classification of obesity: what is a normal BMI?

The World Health Organization in 1995 [1] accepted the body mass index (BMI) as the appropriate method for crudely assessing degrees of underweight and overweight. They took the 18.5 limit for distinguishing normal from underweight following a series of earlier international analyses of the capacity for work and propensity to infections which came with progressively lower BMIs [2]. The upper normal limit of BMI 25 was also taken from earlier analyses which had originally been based on mortality statistics from the US [3, 4], but it was not until the WHO Expert Consultation on obesity in 1997 that the implications of overweight and obesity were accepted by WHO as a global problem [5]. At that time there was considerable discussion about the appropriateness of the choice of BMI as the index because in the US it was accepted by doctors that there were so many heavy adults that perhaps one should only consider an individual overweight if their BMI was 28 or more. The Japanese representative, however, was pressing for a lower BMI cut-off and by the year 2000 the WPRO branch of WHO had accepted an upper cut-off of 23 [6]; above this value the risks of diabetes and hypertension (HT) in particular were unacceptably high. This was generally accepted following the WHO Singapore meeting in 2002 [7], but has not been applied on a regional basis although numerous national bodies in Asia take the lower BMI criteria for granted.

Mortality as the arbiter of cut-offs

The choice of cut-off point implies that there is a distinct increase in mortality and/or risk of disability at levels above this value, but it was recognized many years ago that the risk of diabetes and HT increased progressively from BMIs of about 20. We now recognize, using the Disability Adjusted Life Years [8] system of assessing the impact of a disease that the disease's disability is usually quantitatively far more important than the number of years of life lost prematurely. Yet the original data for choosing appropriate

'normal' weights were based on US insurance statistics relating to differential death rates. This clear hard end-point for specifying appropriate 'normal' BMIs was also based on data in affluent societies because there were no comparable data available for Asia, Africa, the Middle East or Latin America. In those regions life expectancy was much shorter with high prevalences of infectious diseases.

More recently mortality data from the US, involving nationally representative data from the NHANES series of surveys, have produced controversial findings using new modelling techniques with the individuals who were monitored in the first three surveys being assessed for whether or not they had died 10–20 years later [9]. The BMIs associated with the lowest death rates were about 25–28 and the risk of death associated with high BMIs seemed to be falling on a secular basis. These findings are intriguing but need to be taken with some caution. We showed many years ago that identifying the mortality effects of overweight (BMIs 25–29.9) required many years of follow-up with very substantial numbers of subjects [10] and, in practice, the apparent secular decline in death rates also involved progressively shorter periods of follow-up in US analyses. The data involved about 10 000–15 000 subjects in each of the three surveys. The analysis has also been criticized for not fully allowing for two forms of statistical bias: (i) the reverse causation phenomenon whereby some of those with lower weights are already ill and (ii) the subjects' progressive increase in weight during the period of observation [11, 12]. Once these corrections were made, then overweight was associated with a clear increase in mortality. The British Whitehall study, with over 18 000 men but followed up for up to 35 years, showed that all cause and ischaemic heart disease mortality rates – but not stroke death rates – were increased in the overweight group [13]. Another recent integrated analysis of 33 cohorts, followed for an average of 7 years from the Asia-Pacific region and involving nearly a third of a million adults, showed that the risk of fatal and nonfatal cardiovascular disease, particularly ischaemic stroke and ischaemic heart disease, increases progressively from a BMI of 20; however, the impact on haemorrhagic stroke

was not evident until BMIs were in the region of 30 [14]. As in many of these studies, the first 3 years of follow-up in all the collaborating cohorts had to be discarded because those who were already ill had lower BMIs and suffered early deaths so without this adjustment the curves were J-shaped. Similarly, when over half a million US subjects were followed for up to 10 years, it is evident that there was a remarkably rapid increase in death rates from a BMI (albeit self-reported) of 25 [15] in those who had never smoked. This relationship was more evident when the follow-up periods were long and the avoidance of the marked confounding effects of smoking was important.

An optimum BMI?

One also has to recognize that all the choices of cut-offs – whether for designating HT, glucose intolerance or hyperlipidaemia – are very arbitrary. It is well recognized that the optimum systolic blood pressure should be about 115 mmHg and Chinese data many years ago showed that the incidence of coronary heart disease (CHD) was proportional to the blood cholesterol concentration down to levels which matched those observed in rural Africa. Thus, the optimum levels chosen in the WHO Millennium analyses, were for systolic blood pressure 115 mmHg, total blood cholesterol 3.8 mmol L⁻¹ and, in our case, a BMI of 21 [16]. This choice of BMI was based on our finding the mean population value which would limit the likelihood of individuals being either underweight or overweight. Indeed, there are substantial numbers of cohort studies showing that, for individuals, the lowest risk of diabetes, HT and other conditions may be at BMIs <21.

Cut-off points based on sensitivity and specificity analyses

This has become a popular approach where the target is to work out which BMI should be chosen on the basis of the receiver operating characteristics (ROC) approach [7]. This balances the need for sensitivity in screening, i.e. picking up the maximum percentage of cases with complications of weight gain such as HT

or diabetes (DM), balanced by the need to screen as few people as possible without the condition (the specificity of the approach). The original choice of BMI 23 for Asians by the WHO/IASO/IOTF meeting in Hong Kong was based on a preliminary pragmatic inspection of data relating BMI to DM and HT rates [6]. Later, however, it became conventional to use the ROC analysis for identifying cut-off points, e.g. for designating the 'normal' BMI as <23.9 in China [17]. More recently a far more extensive analysis has been conducted involving over 16 000 adults from Asia which showed higher diabetes and HT prevalences at each increment in BMI. For example, at a BMI of 24 kg m⁻² the proportion of men with diabetes was 5% in Asians compared with 2% in Caucasians, and in women the corresponding values were 5% and 1% respectively. The optimum discrimination points for HT and diabetes were a BMI of 24 and a waist value of 85 cm in men and 80 cm in women [18].

Clearly, from a national policy perspective, there is a more democratic imperative, i.e. to have a system that maximizes the number of people detected with DM and HT without having to screen everybody. In Mexico, the government had given a commitment that at least 80% of people with undiagnosed type 2 diabetes and HT would be identified and treated. We then found that on this basis screening everybody with a BMI of ≥ 24 would identify >80% of DM and HT affected adults, with 65–70% of the population needing to be screened [19]. However, by using a waist circumference (WC) of ≥ 90 cm, only 55% of women and 65% of men would need assessing. To identify >90% of cases needed a WC cut-off of 83 cm. This compares with the use of a WC of ≥ 95 cm for both men and women, based on ROC analyses, which would have minimized the effort but missed 35–40% of cases. Furthermore, if the strategy is to communicate with the public and provide them with a suitable optimum measure, then it is clear that in Mexico a waist value of <83 cm could be used as a very reasonable way of ensuring that both men and women know the waist value that minimizes risk. Similar analyses have not as yet been conducted elsewhere.

Table 1 Clinical relevant values for screening and potential clinical use in different population groups but often based on different criteria

	Waist (cm)			BMI (kg m ⁻²)		
	WHO (Caucasian)	Asian	Hispanic	WHO (Caucasian)	Asian	Hispanic
Men	≥94 102	≥90	≥90	≥25	≥23	≥24
Women	≥80 88	≥80	≥90	≥25	≥23	≥24

Adapted from Sanchez-Castillo [21].

On this basis one can derive a variety of measures of both BMI and WC which can be used in a clinical screening context so that any propensity to HT and type 2 diabetes can be considered. Table 1 shows what seem currently to be appropriate guidelines, recognizing that the WHO and Asian criteria are based on different concepts which have been dealt with in more detail elsewhere [20, 21].

Asian and Hispanic sensitivities to weight gain

There was, of course, pre-existing evidence that at equivalent BMIs (and indeed WCs) diabetes was far more prevalent in Asia and was exacerbated by weight gain [22]. Subsequent comparisons undertaken for the Millennium burden of disease analyses also showed that Scandinavian adults had an absolute risk of diabetes which was a third or half of that found in Japanese, both studies being representative and using fasting blood glucose as the criterion for comparing the two countries [23]. The data also showed that the gradient of risk was the same as the BMI increased in the two countries, so clearly there must be another factor which confers a greater absolute risk of diabetes in the Japanese. This might quite reasonably be considered as genetic given the well recognized, and newly emphasized, criteria established for the molecular basis of genetic sensitivity to the development of type 2 diabetes [24]. However, before jumping to the conclusion that this is the sole factor, one also needs to consider the increasing evidence that early foetal and childhood 'programming' can also increase the likelihood of developing diabetes. Thus, Barker in his original studies in Hertfordshire, England found that small birth weight babies had a much greater risk of developing both abdominal obesity and an enhanced risk of diabetes and HT in late middle age. Heavy babies born at that time had a fifth of the risk of

diabetes compared with that in low weight babies [25]. Whilst birth weight was taken as a simple crude index of probable maternal nutritional insufficiency, there were other grounds for knowing that women in the working class of the UK were very deprived and on poor diets in the first three decades of the 20th century [26]. Since then there have been a series of reports confirming these relationships between indices of foetal and infant growth and later metabolic problems, for example, in Finland [27] and also in China [28]. This concept of intergenerational effects affecting almost all societies at one time is illustrated in Fig. 1.

Early determinants of insulin resistance and possible hypertension

India looks as though it has perhaps the greatest amplification of risk as weight increases. Indians have a marked tendency to develop abdominal obesity which Yudkin and his colleagues have ascribed to a variety of environmental factors including the cytokine inflow from chronic gut infestation and infection in an environment with extremely poor standards of clean water and sanitation [30]. More recently, Yajnik has been emphasizing the importance of the dietary contributors in pregnancy and has highlighted the critical role of the folate/vitamin B12 contributions to both one carbon pool metabolism – so essential for nucleic acid synthesis – and to the possibility that changes in methylation capacity may compromise the epigenetic methylation control of the promoters of genes controlling differentiation at a very critical stage of the Indian foetus's development. In practice >70% of Indians, by virtue of being vegetarian, are B12 deficient so the current practice of providing Indian mothers with substantial doses of folic acid during pregnancy to counter the widespread coexisting folate deficiency in India

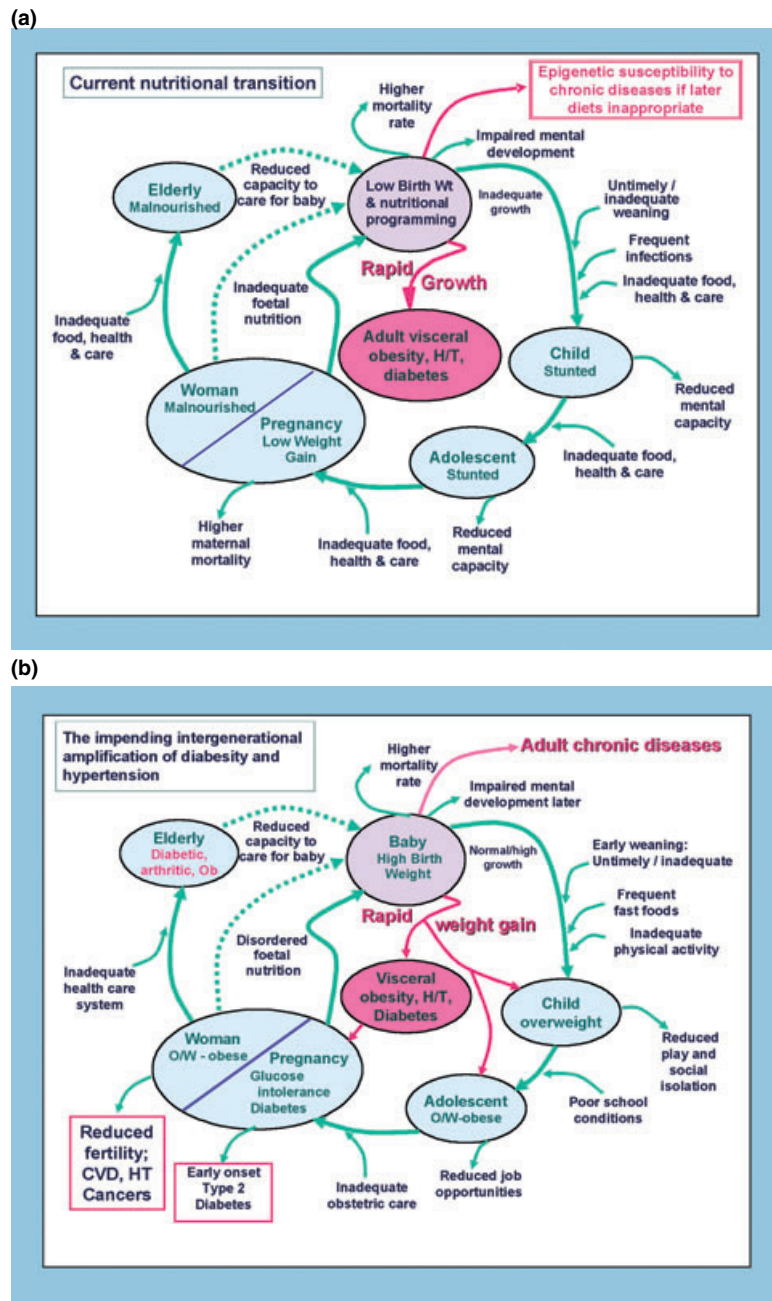


Fig. 1 The intergenerational lifecycle: the proposed causal links of the current international nutritional transition (a) but already fat adolescents are entering pregnancy to produce diabetes and hypertension prone children (b). Adapted from James *et al.* [29].

may distort the control of one carbon pool metabolism and lead to even greater insulin resistance than that documented with B12 deficiency alone because the higher the folate and the lower the B12, the smaller

and fatter the babies and the greater the insulin resistance [31]. There is also a documented link between the high maternal plasma homocysteine levels and low folate, low B12 levels and low birth weight [32].

This issue of B12 and folate deficiency might be considered an unusual Indian feature but careful scrutiny of data from across the world shows that folate deficiency is exceptionally common, as is B12 deficiency, in many low- and middle-income countries – usually described as the developing world. Thus, it is not uncommon to find 50% of adults with modest folate deficiency and a quarter or more of the population with low vitamin B12 levels [33].

These nutritional factors seem to be involved in the early programming of organ size, metabolic flexibility and, potentially, the growth of the child, but the development of excess weight gain after birth also depends on the prevailing environmental conditions. Breast feeding seems to protect a child from subsequent obesity but this is often been ascribed to the concomitant socio-economic circumstances of the family. More recent studies, however, do suggest that breast feeding genuinely protects [34] and this may, in part, be explained by the fact that mothers have little understanding of how much energy their child is obtaining from breast milk as the amount taken is highly dependent on the child's own neural control of appetite. This mechanism may therefore be fine tuned during the early months of life when breast feeding allows the controls to be established on an individual basis and in keeping with the child's own lean tissue mass, differential organ size and energetic efficiency. Bottle-feeding, however, may unwittingly lead to a readjustment in these controls which allow greater weight gain as the children may inadvertently be overfed by an anxious mother seeking to satisfy her new baby. Then in our current environment many children, even if originally breast fed, start gaining excessive weight once they are 2–4 years of age as environmental factors mean when they are exposed to very inappropriate foods and eating patterns.

A public health perspective

Although the epidemic of obesity really started to increase markedly in the 1980s it is only since 1997 that WHO and many national governments have recognized the importance of obesity as a major public health problem affecting both the developed and the

developing world. WHO regional offices have also been involved in taking a series of initiatives to cope with the epidemic in their regions. The WHO European Office and the European Ministries of Health have gone on to agree in late November 2006 a charter for tackling obesity. This, of course, depends not only on establishing the basis of the epidemic, but also the most effective preventive and management measures.

The serial increasing trends in obesity prevalence that were evident in most Western countries, especially the USA, from about the beginning of the 1980s affected not only adults, but also children. The documentation in adults was much easier as the use of BMI was readily accepted, but the paediatric world, whilst interested in unusual genetic problems associated with obesity, only accepted that this was a public health problem at the beginning of this Millennium when the International Obesity TaskForce had established a reasonable scheme for linking the children's cut-offs for BMI (which changes by age and differentially by sex) with the accepted adult cut-off points of 25 and 30 [35].

Since then we have documented the obesity epidemic in adults and children and shown that this seems to be rising relentlessly (Fig. 2), with 20% of all adults in most European countries already obese and with higher rates often found in the Southern, Central and Eastern European nations. Similarly, the Middle East has exceptionally high obesity levels and is also the region with the highest national prevalence rates of type 2 diabetes in the world. Latin America is similarly affected with the poor Caribbean Islands now overwhelmed with a medical burden of premature diabetes and cardiovascular disease. Asian and African countries are also showing dramatic changes in both physical activity (PA) and diet associated with very rapid increases in obesity prevalence and its associated type 2 diabetes.

Physical inactivity

The obvious thermodynamic principles of energy balance require that one recognizes the influence of both

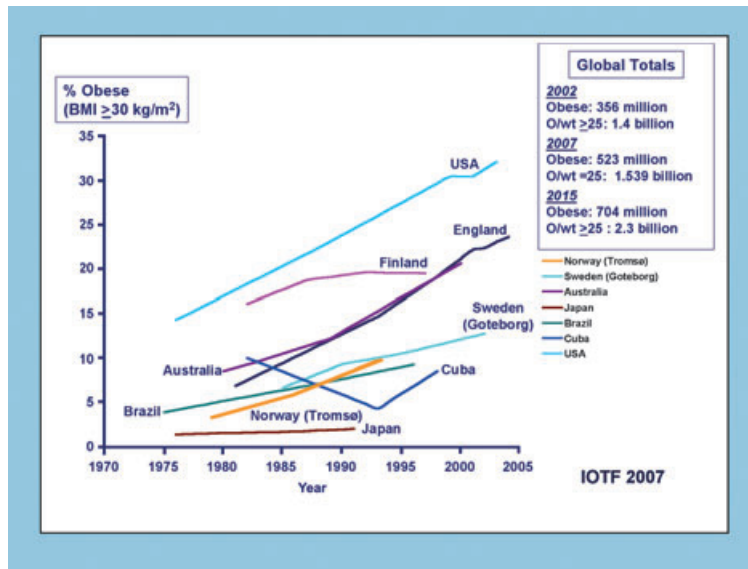


Fig. 2 Current analyses of the escalating obesity rates in different countries: only Cuba during the financial and food crisis showed a decrease but Finland is doing better than other countries in modifying the problem.

dietary change and the reduction in PA. The collapse in the demand for physical exertion came with cheaper cars for personal transport and multiple mechanical and electrical aids to remove the physical demands in the home and at work. Then, with the advent of computers and television, it is now clear that one can earn an excellent wage and have enjoyable leisure with virtually no physical exertion.

The evidence for current trends in PA varies, but most affluent countries have noted relatively stable rates or slight declines in leisure time physical activity (LTPA) over recent decades [36] and in general the rates of sedentariness, defined as an absence of LTPA, have remained rather stable. Of more importance to total energy expenditure has been the decline in other domains of PA. Rates of heavy occupational PA have declined dramatically, with more than a 50% reduction in 'heavy work' in Norwegian adults since the mid-1980s, and associated increases in sedentary work [37]. Other countries have noted marked increases in car usage and other motorized transport, at the expense of more active commuting through walking or cycling trips [38]. One study even reported a 6% increase in the risk of obesity for every hour spent commuting by car each day [39]. Limited data are available on domestic settings, but with increases in technologically sophisticated labour-saving devices,

and reduced time to prepare meals and carry out household tasks (predominantly through increases in dual adult working households), it seems that energy expenditure on domestic tasks and yard/garden work is likely to have reduced as well [36].

For some European countries, the increase in obesity was delayed by up to a decade, notably in the Netherlands and Scandinavian countries [40]. This might have been due to high rates of active commuting, especially cycling, as nutritional differences are not apparent compared with several other western European countries [41]. A few countries have demonstrated increasing trends in LTPA, over more than a decade. In particular, Finland and Canada have increasing LTPA trends over 25 years, and Singapore over about 10 years [42, 43]. Note that these comparisons are reliable within-country, but are not comparable between-countries because of different PA measures used [44]. Despite increases in LTPA, all of these countries have observed increases in obesity rates that were similar to demographically matched countries which did not have increases in LTPA [43, 45–47]. For Finnish adults, the rates of increases in obesity were marginally but not substantially faster amongst the inactive [46]. Similarly, Anderssen [37] showed that (leisure time defined) active and inactive Norwegians have gained weight at roughly similar

rates since 1990. This paradox is likely to mean that, in spite of LTPA increases, obesity rates increase because of overwhelming increases in energy intake, or the obesity must have developed in association with substantial falls in energy output for tasks other than leisure time activities.

Evidence for the transformation of our diets is easier to document but the mistake made by doctors, scientists and policy makers is to assume that we can measure the diet with sufficient accuracy to discern imbalance which, on average, amounts to 10–20 kcal day⁻¹, i.e. <1% of normal daily intake and expenditure. Given the well recognized substantial variation in energy intake from day to day and week to week, and also the wide variation in our usual pattern of PA, the relative stability of energy balance reinforces the experimental evidence that we have surprisingly good control of our energy balance under 'normal' circumstances. There are, in fact, a complex, multifaceted series of mechanisms which are designed to regulate overall intake by short-term meal-based factors affecting satiety, snacking and inter-meal intervals and also medium as well as long-term regulatory processes. These mechanisms reinforce the primary need to prevent weight loss and are multiple and powerful, whereas those which prevent weight gain are less robust. Nevertheless, under the usual environmental conditions before 1980 children and adults on average were able to maintain their energy balance reasonably well unless they became ill and anorexic. At that time it also seems clear that there was sufficient demand for physical work both in the home and in the usual range of occupations for people's appetite regulatory centres to be repeatedly operating to ensure that sufficient food was eaten to satisfy energy needs.

Assessing the basis of energy imbalance and resistance to slimming

One is fortunate if one can be confident of measuring energy intake to within 500 kcal day⁻¹ of the true figure. Therefore, assessing which food group or dietary practice is the prime driver for what is often assumed to be a very substantial increase in energy intake

usually depends on characterizing the type of diet associated with weight gain and then attempting to do intervention trials for a minimum of 6–12 months where one has to overcome the normal reaction of people to continue previous food habits. Unfortunately, in addition to these social issues there is now very good evidence that as weight gain occurs, particularly in older adults, there is a 'resetting' of the regulatory system so that weight loss is resisted. The obese then demonstrate acute hormonal, hypothalamic-regulated responses involving both the thyroidal axis and the autonomic nervous system which simulate the response to semi-starvation within 2–4 days, even though the obese may have 0.25–1 million extra kilocalorie stored! Furthermore, this adaptation seems to be a progressive process because the initial average daily energy imbalance is very small but the weight gain progresses when it should stop as the extra weight, with its intrinsically higher basal and movement costs, balances the additional intake. This continuing weight gain then means that in due course the additional 10 kg extra weight demands a permanent extra daily intake of 200–300 kcal day⁻¹. This adaptive mechanism does not occur after acutely overfeeding young adults because it has been shown that they spontaneously return to their previous body weights, but this is not seen when older adults are acutely overfed [48, 49]. This adaptive response, or failure to rectify excess fat accumulation, also seems to apply when weight gain is more gradual. If diligent patients follow their instructions strictly and manage to bring their weight down to normal despite all the intense brain signalling to return to their previous increased weight, they are then confronted with the need to follow obsessively a rigorous programme for monitoring their food intake and purchasing habits. In addition, they usually need to deliberately engage in a 2000–3000 kcal week⁻¹ exercise regime to avoid regaining the lost weight [50].

These adaptive systems therefore seem to explain why the obesity epidemic continues to increase despite the desire of so many adults to lose their excess weight. This indicates a need for prevention to become a very high priority. The dilemma is illustrated in Fig. 3

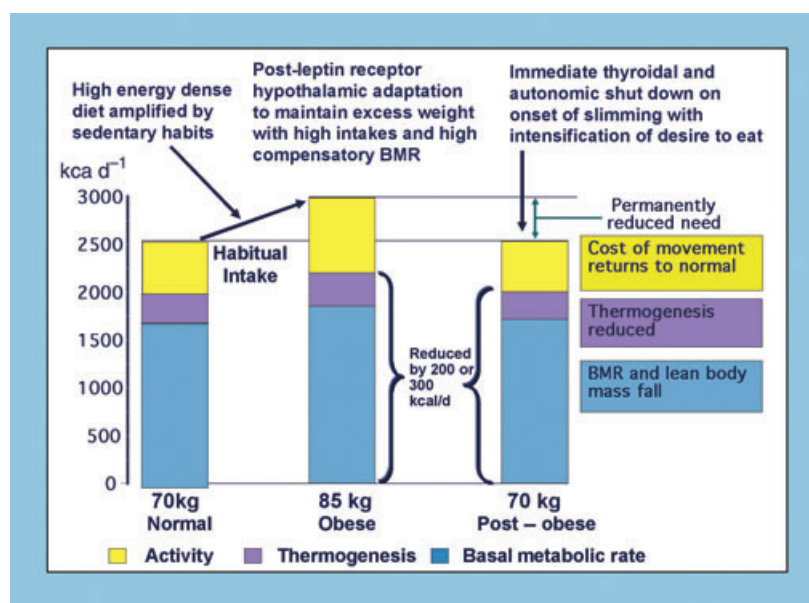


Fig. 3 The sequence of energetic and physiological adaptations to weight gain and subsequent slimming explaining the frequency of weight regain.

The roots of the problem

So how do we explain the start of the epidemic? It now seems clear that with the removal of the need for PA to earn our living, the prevailing pressure on our brain regulatory systems has been to attempt to limit intake. It seems reasonable to accept that we have already moved away from the fabled large meals of our ancestors. Indeed, within our life time, in relatively sophisticated environments the meals and portions served in restaurants – at least in Europe – have become smaller because that is what consumers seem to want. Thus, data from national food surveys in the UK showed a progressive fall in consumption even when allowances were made for the greater amount of food now eaten outside the home. Similarly, there are many people now who do not bother with breakfast and this may well be a behavioural adaptation to a brain system which is trying to stop us eating so much. However, there have been remarkable changes in our food purchasing habits with an ever-increasing tendency to eat outside the home or buy ready-to-eat or preprepared meals rather than cook with basic ingredients. This tendency has been amplified by more women working outside the home and increasing number of single households. Cooking skills have tended to play a lesser part in school curricula and

children are less likely to have the example of cooking skills within the home. This means that we have become far more dependent on manufactured, processed foods and make decisions based on the information provided by the food industry.

The response of the food industry

Since the early 1990s there have been major developments throughout the food manufacturing, processing and retail sectors, creating one of the most sophisticated and powerful marketing forces in the Western world. Products have been fashioned and formulated in response to extensive feedback from consumer taste and market research panels, creating an ever-expanding market for novel foods which will appeal to an increasingly sophisticated market as people travel and experience new flavours and foods.

Unfortunately consumer taste panels have, unwittingly, tended to endorse energy dense foods full of fat, sugar, salt and artificial flavours which appeal to the selective taste receptors for salt, sugar and essential fatty acids, as well as 'umami'. The latter taste characteristically responds to monosodium glutamate and to cooked meats and other high-protein foods; it

is sometimes considered to be a fifth basic taste along with sweet, sour, salt and bitter. These receptors were set by the primeval drive for essential nutrients in our evolutionary past when these commodities were precious and scarce in our African environment.

Producing energy dense and concentrated foods has also been helped by refining starches and removing fibre, thereby reducing the water-holding properties and physical bulk of foods. Thus, a huge range of highly energy dense products has emerged, providing appetizing, quick to cook and easy to eat foods which dominate supermarket shelves to the detriment of fresh products which require a degree of purchasing knowledge and cooking skills to fashion meals of a comparable standard.

Numerous double-blind controlled trials have now shown that manipulating the energy density of meals does not immediately impact on the amount of food eaten so that the energy-rich meals are eaten in similar amounts. This can soon lead to what is termed 'passive over-consumption'. Furthermore, marketing experts have discovered that they can change the food and drink culture. For example, in the US they encouraged young people to believe that it was 'cool' to carry coffee around in the street, and then assorted foods to go with it. Regular meals soon gave way to 'grazing' and a huge additional marketing opportunity was created to sell immediately available products in easy to access packages accompanied by a variety of soft drinks and sophisticated variants of coffee, chocolate and tea. This in turn means that the brain regulatory system is having an even bigger struggle to limit intake; no longer is it trying to operate on the basis of a three or four regular meals a day, but it has to try to counteract the incessant pressure on people to buy immediately available, heavily marketed and attractive commodities for immediate gratification. Now, with the neurosis about obesity, it is common in Western societies to see women walking around clutching a bottle of spring water which is deemed to be sophisticated but also implies that they will rapidly become dehydrated without a constant supply of liquid.

Industry and governments have known for a long time that to change people's food habits requires attention to three specific issues:

- 1 food pricing;
- 2 food availability (both purchasing power and ease of access to food outlets) and
- 3 the marketing of foods and drinks.

These lessons were learned in the UK and Scandinavia during the Second World War when food was scarce and there was a need to ensure that people were fed properly. Post-War, fundamental decisions were taken both in the West, and then in the Soviet Union, to ensure national self-sufficiency in food; preference was given to meat, butter, milk, fat and sugar production, with price restrictions and inducements to ensure there would be sufficient protein and energy for the whole population.

The result of all this was 50 years of massive agricultural subsidies whilst doctors and nutritionists advocated an ill-defined 'balanced diet', the concern about vitamin deficiencies in Western societies apparently no longer being a dominant consideration. So the value of fruit and vegetables was downgraded and they were the least subsidized and supported commodities.

Thus, agricultural policies and the many and varied industries contributing to all aspects of the food chain, became responsible for the progressive cheapening of meat, butter, oils, fats and sugars, whilst the relative price of fruit and vegetables climbed. Given this perspective it is little wonder that, as the epidemic of cardiovascular diseases developed during the 1950s and 1960s, governments concentrated on 'health education' messages, despite clear evidence showing that this approach had failed dismally in the past in the face of the huge commercial and marketing pressures on people not to comply. Indeed, failure is more or less guaranteed when one considers that government expenditure on health education, and nutrition education in particular, is about 1% or less of the marketing budgets of the combined food manufacturing, fast food restaurant and supermarket sectors.

Then there was a response to the demand for lower saturated fat intakes to reduce heart disease and stroke rates, combined with a campaign to reduce smoking. Smoking restrictions developed slowly: progressive taxes raised the price; measures were introduced to restrict the sales outlets, particularly to children, and a ban put on the marketing of tobacco products. Here, again we see the effects of the trio of forces for population behavioural change – price, availability and marketing.

The industry response to the anti-saturated fat crusade was to sell their products as low fat or low saturated fats. They also introduced polyunsaturated margarines and other products. Half the reduction in saturated fat intake came from consumers choosing to purchase low fat milks and spreads, and the other half from manufacturers realizing they could still make foods palatable with polyunsaturated fats and at the same time claim health benefits! The subsequent fall of CHDs and strokes in the Northern European countries which have developed multiple sustained governmental interventions throughout the societal system has been impressive.

Obesity prevention: optimum fat intakes and physical activity

Tackling obesity, rather than the other chronic diseases, presents greater difficulty because there is the fundamental need to reduce energy intake rather than simply manipulate the type of fat. Nevertheless, the strategies which were adopted in Finland to increase fruit and vegetable intake whilst simultaneously reducing total fat and salt intakes not only helped to reduce the average blood pressure, but also probably contributed substantially to limiting the rise in obesity rates which were beginning to rise rapidly before the fat intake dropped below 35% of energy and the average vegetable intake trebled. Thus, although not presented as such, there would have been an appreciable fall in the average energy density of the diet although that would not be sufficient to combat the intrinsic adaptive systems for preserving body weights.

The obesity report produced by the WHO from its 1997 consultation considered 20–25% to be the maximum average intake which would limit obesity, but the dominant policies are still those relating to cardiovascular disease developed by Geoffrey Rose and Henry Blackburn for WHO in 1984. They specified a 15–30% range in total fat intakes because the Chinese and Japanese, with negligible CHD, diabetes and obesity in the 1970s, were consuming on average 14% fat, but this was totally unrealistic for the US and Northern European populations where intakes were well over 40%. So to choose a 30% figure was radical and a response to the need to reduce saturated fat intakes by simply reducing total fat. Ancel Keys' Seven Country Study observations that fat might be related to the BMIs of his samples but not to CHD because the Greeks, on a high olive oil diet, had a low saturated fat intake at that time. The subjects also happened to be incredibly physically active: the selected shepherds in Crete were walking and climbing mountains all day long so this is hardly a realistic basis for suggesting that fat intakes do not matter.

It is undoubtedly true that a fat intake with negligible saturated fat is compatible with a healthy body weight and a low rate of type 2 diabetes, but this demands substantial rates of energy expenditure – probably requiring exercise at a rate of at least 2500 kcal week⁻¹ which is a totally unrealistic population strategy for all men, women and children of all ages. In our preliminary assessment, based on detailed studies of postobese adults, we realized that a physical activity level (PAL) of >1.8 would be needed for these individuals to cope in a Westernized environment and in addition there would still be a need to make major changes to their diet. In reality, the PAL value of 1.8 would mean individuals walking relatively briskly for an additional 60–90 min every day of the week, i.e. with >10 000 steps recorded on a pedometer. Thus as explained above, leisure time activity is not an appropriate strategy to promote as a single measure for combating the problems of obesity and we need a fundamental shift in thinking. We need to reshape our built environment to encourage spontaneous walking in safe surroundings; this will require national

planning and country-wide, interdisciplinary cooperation. We have currently spent billions of euros on promoting motor car use and limiting public transport on trains, buses and trams. This has led to a very substantial fall in the rates of spontaneous cycling and walking because in most countries it is no longer safe to do so or as in the US cities almost physically impossible. Indeed, in China, the transformation from the population using bicycles as a routine to the current obsession with emulating US car use is probably the most startling national change in any 5-year period except in times of war and other crises.

Integrated public transport systems are a key to societal changes in activity. Thus, considerable distances may be spontaneously walked if the environment is interesting, safe and well lit after dark. Pedestrian-only walkways are particularly conducive to family activity where parents can relax and move around more freely with youngsters. Urban car use can be deterred through congestion charging and access permits, but promoting new, and restoring existing, green open spaces and safe play and leisure areas in dense urban development and restoration is a far greater challenge.

The UK Foresight report on obesity

The UK government's Chief Scientist has spent the last 2 years attempting to explain why the population is becoming progressively more obese despite the widespread desire to slim [51]. Table 2 sets out a summary of the findings; the Foresight report also came up with a crude summary of the relative strength of the different forces affecting the development of obesity in the UK. As noted above the problem is seen to be a 'passive' normal reaction of the human body to the prevailing obesogenic environment so that everybody unless exceptionally endowed genetically is bound to put on weight. Thus, this is a socio-economic problem where the environmental circumstances overwhelm the normal physiological controls. The problem is so severe that an overwhelming medical crisis is inevitable unless the problem is seen as a huge multidimensional issue which requires everybody to change their policies and practice. This means that it is no longer reasonable to simply

Table 2 A summary of the findings of the UK's Foresight analysis of the obesity problem [51]

1. The epidemic of 'passive obesity' is a normal response of our innate biology when confronted with modern living 'obesogenic' environments so everybody except the genetically very resistant are guaranteed to put on weight except in unusual social circumstances
2. Increasing obesity rates are guaranteed under current circumstances: by 2050 60% of men and 40% of women will be clinically obese at a very conservative cost of £45.5 billion year⁻¹
3. A societal approach is needed: the epidemic cannot be prevented by individual action alone
4. The magnitude of change needed is greater than anything tried so far and must involve at multiple levels of societal change: personal, family, community and national
5. Obesity is similar to the climate change challenge. It requires partnership between government, science, business and civil society; it reflects a 'market failure'

specify the individual changes needed but now every branch of society has to be involved if we are to avoid an overwhelming financial and societal burden. The challenge is so great that it is likened to climate change and is a clear indication of 'market failure'.

The challenge of prevention

As general initiatives on obesity have only just started we should not be surprised by the very limited information that we have on practical community developments which have been shown to be successful. Developing public health policies to combat obesity is also a much more complex process than we originally anticipated and there are all sorts of political pressures to cope with. Thus in many countries, e.g. the UK and Sweden, strategies and policies have been devised but not implemented, primarily because politicians have not yet taken on board the Foresight perspective that obesity is an inevitable passive response of human beings to what we now term a 'toxic' or 'obesogenic' environment. This broader perspective is dependent on recognizing that community intervention projects are much more difficult to conduct than simple drug trials. They also involve many different factors and nonmedical sectors which are hard to

evaluate in a rigorous, experimental way. Therefore, as with most other aspects of Government policy-making, e.g. social and economic, one has to work on the basis of understanding the causes of the problem, evaluate the primary drivers and then identify suitable options for changing the environment. Politicians readily understand these processes in other spheres but are constantly hearing from critical medical establishment figures that the success of any scheme is a matter of conjecture. In other policy areas, with less science to support the proposals, there is more consistent backing from experts of policy initiatives because of the cultural acceptance of the legitimacy for initiating change supported by previous analyses or models based on currently accepted theories. Politicians are also inhibited by the power of the food producers, retailers and restaurant industries who, with agriculture, are the biggest industrial players in most countries. They have also been influenced for decades by the motor car industry and other major engineering and now computer manufacturers who have immense political power and seek to operate in as free a market as possible. They are, understandably, primarily concerned with short-term profits and it is only when public opinion changes and forces through new regulatory approaches, or where business sees a new market opportunity, that they will act in concert with those concerned with public health.

Immediate measures needed

Food labelling

For years, food labelling has been a notoriously complicated issue with a long-standing debate about its role and impact on food purchasing. It is now accepted that many of the existing labelling schemes are confusing and often misleading so there is an urgent need for a new informative, and preferably universally accepted, format that can be easily understood by all consumers. There is evidence to suggest that the use of labels which identify 'good' foods, e.g. with a tick (Australia), or with a green key (Sweden) can be used by intelligent, informed and motivated consumers, but it is generally recognized that this system is not reinforced by government health

injunctions to limit the purchase of the foods without a tick or green key. This system is essentially therefore a political compromise and gives only a small part of the picture of food purchasing options. If ticks and keys specify what is in effect a 'good' food then a government and the medical profession has a responsibility to insist on labelling 'bad' foods too!

We have therefore advocated a universal traffic light nutritional signposting standard, based on WHO goals for fats, sugars and salt [52]. This has been shown to be preferred by most consumers and can be more easily understood by the less well-educated purchaser. Furthermore, if producers manipulate the composition of a processed food so that it has a series of red circles denoting that it is high in energy, total fat, saturated fats, sugars and salt, it will be rejected in favour of an equivalent product which has been nutritionally manipulated to allow several more green and orange circles. One such system has been developed by the UK's Food Standards Agency [53] and tried by the UK supermarket J. Sainsbury plc with remarkable evidence of positive consumer responses. This uniform approach needs to be applied to all food packaging, supermarket shelves, canteens and restaurant menus. It is likely to be the most effective means to inform and influence consumer behaviour through genuine, as distinct from spurious, 'informed choice'.

Marketing

Children clearly need to be protected from the pervasive effects of food marketing which is now accepted to have a powerful and deleterious effect on their knowledge, food preferences, their purchasing habits, as well as those of their parents, and their food intakes. The protection needs to apply to all forms of marketing, including Internet, individually targeted 'viral' marketing via mobile phone networks, in-school promotions as well as existing television marketing. The evidence for the deleterious effects is compelling [54, 55] for preteenage years but the precautionary principle should apply to teenagers, who are particularly vulnerable to the impact of marketing psychology and media influences on adolescent behaviour and development. It should be emphasized

that the Convention on the Rights of the Child specifies children as those under 18 years. In addition, special care is needed in relation to the presumed benefits of social marketing, which does not take into account the poor understanding of the less educated and more vulnerable members of society and is unlikely to be able to compete with the huge marketing investment of the private sector. It should become a social norm not to have any industrially driven marketing to children. Governments should also adopt measures to monitor marketing restrictions so that the voluntary or regulated restrictions can be scrutinized.

Nutritional quality control

All government and other premises assigned for children, e.g. nurseries and schools and for workers, e.g. in government run or supported facilities, should apply mandatory nutritional and activity standards to all their institutions. Thus, for example, it is ludicrous to see inappropriate fast food, confectionary and soft drinks on sale in hospital premises when these contribute substantially to the very conditions overwhelming the hospital work load. Public procurement policies should also support nutritional standards, with an emphasis on local fresh food supplies. Special arrangements are also needed to encourage PA.

These measures need not only apply to government premises. Businesses could well take a lead in promoting the most effective measures for their own financial benefit in terms of their workers' time off work for hospital visits and sickness as well as for general societal benefit.

Pricing

Influencing food pricing is going to be difficult because we need to address the imbalance induced by the cumulative impact of a half century of misdirected state funding which has nurtured the overproduction, and therefore the excessive marketing of, foods that contribute to harmful diets. Indeed, price intervention mechanisms and production controls have hindered the availability of cheaper fruit and

vegetables, needed to promote quality standards for healthy diets. Financial structures need to be revised to favour increased consumption of fruit and vegetables and discourage over-consumption of foods high in fats and sugars and high caloric beverages. Revenue neutral changes should be introduced to support price reductions and thereby provide incentives to consume nutritionally healthier foods and price increases on food and drink high in fat, sugar and salt – the 'HFSS' foods. So financial and social policies need to be modernized to promote a new health agenda. So far the free market has only partially been applied and has helped to promote inappropriate industries and a huge public health problem. However, it is totally reasonable to limit the total free market until we have induced changes which industrially favour a marked improvement in food patterns. Such approaches are essential if the agricultural sector is to provide those foods needed at affordable prices for good health.

This remedial step may not seem straightforward but analyses undertaken over a decade ago showed how the patterns of food production would need to change if everyone consumed the optimum diet which has been set out for the last 15 years by consensus WHO groups. In practice, the aggregate change demanded of the US agricultural sector would be relatively small, requiring a net increase of only 5–6 million acres of crops, or about 2% of total cropland planted in the early 1990s [56].

Availability

Local as well as central governments should contribute by restricting the availability of fast food/soft drink outlets in their own premises and indeed in urban environments in ways analogous to alcohol and smoking restrictions. Restrictions need to apply particularly to outlets near schools. The increased availability of fruit and vegetables should be encouraged by suitable planning measures which are in regular use in many countries. This requires, however, a coherent central group or agency to coordinate and maintain pressure and with a monitoring role of these local government changes.

Conclusions

The epidemiology of obesity can be considered intriguing from many points of view. Seeking to understand the propensity to type 2 diabetes after slow intra-uterine and infantile growth, as observed in British, Finnish, Dutch, Chinese and many other communities, can contribute not only to the development of techniques for monitoring the control of the one carbon pool *in vivo*, but also the way in which epigenetic modulation of gene expression and organ development is determined by direct substrate availability or indirectly through different signalling pathways. Body compositional developments are also in need of greater clarification in different populations as well as the genetic propensity, if any, for different ethnic groups to develop obesity and diabetes. In a broader epidemiological context we still do not have coherent data of rates of weight gain on a national and sub-national level in many regions, especially the Middle East, Africa, the Caribbean, Latin America and Asia. Even in Europe we still have only a few countries which systematically monitor the growth of children as well as adults on a regular basis.

We need to develop coherent approaches to delay weight gain, intervene in the millions of overweight subjects and to introduce a system of regular doctor assessment of WCs as part of their alerting system when patients come to see them for any problem and not just for obesity *per se*. Then we need to have coherent plans to tackle the epidemic by medical management in cooperation with other societal groups, including commercial slimming groups.

The biggest challenge, however, is to develop coherent analyses of the broader factors and policies leading to our current toxic environment. We need to try different approaches in different countries and monitor as we go since the escalating pace of the epidemic does not allow us to wait for 10–20 years to gain the sort of detail that we currently expect from drug trials. Developing policies by monitoring the effect of policy initiatives is a well recognized routine of government in other areas so we need to learn different approaches if we are to cope with the huge burden of

chronic disease induced and magnified by excess weight gain which is currently about to swamp our capacity to cope.

Conflict of interest statement

No conflict of interest was declared.

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